surgeons practicing longer. In addition, there appears to an association between personal health habits and the care physicians provide their patients (Schwartz JS et al, Ann Intern Med 1991;114:46-53). Surgeons themselves and the health care system in general both have a stake maintaining a population of healthy and happy surgeons!

Explaining the Decrease in Mortality From Abdominal Aortic Aneurysm Rupture

Anjum A, von Allmen R, Greenhalgh R, et al. Br J Surg 2012;99:637-45.

Conclusion: The reduction in incidence of ruptured AAA (rAAA) since 1997 can largely be attributed to changes in smoking prevalence and increases an elective AAA repair in patients aged ≥75 years.

Summary: Data indicate that prevalence and incidence of AAA is declining and has been doing so since 1999 (Norman PE, J Vasc Surg 2011;53:274-7). Age-standardized mortality rates from AAA also appear to be decreasing (Sandiford P et al, Br J Surg 2011;98:645-51). Factors influencing incidence, prevalence, and mortality of AAA are likely multifactorial reflecting perhaps decreased smoking prevalence, improvements in air quality, and aggressively increasing strategies to protect against cardiovascular risks. All of these factors are also likely to influence population-based incidences of rAAA and rAAA mortality. In this study, the authors assessed the reduction in deaths from rAAA in England and Wales and possible reasons for this reduction. They studied statistics for mortality, hospital admissions, and procedures in England and Wales, beginning in 1997. Data were age-standardized. Trends in smoking, hypertension, and treatment for hypercholesterolemia (statins) with regression coefficients for death available for those aged at least 65 years. Deaths from rAAA avoided in this age group were estimated using the IMPACT equation: deaths avoided = (deaths in index year × risk factor declined) × β-coefficient. From 1997, deaths from rAAA have decreased almost twofold in men. Elective AAA hospital admissions for repair increased only modestly, from 40 to 45 per 1,000,000 population and was attributable almost entirely to more procedures in patients aged >75 years ($P < .001$). Hospital admissions for rAAA declined from 18.6 to 13.5 per 1,000,000 population across all ages. The proportion of patients offered and surviving emergency repair were unchanged. From 1997, death from ruptured AAA in patients aged ≥65 years has fallen from 65.9 to 44.6 per 1,000,000 population. Eight to 11 deaths per 1,000,000 population were estimated to have been avoided by a reduced prevalence of smoking, and a similar number of deaths were also avoided from an increase in the number of elective AAA repairs. Estimates for effects of blood pressure and lipid control were uncertain.

Comment: In accompanying commentary to this article Dr M. Thompson writes, "the epidemic of abdominal aortic aneurysm (AAA) disease may be over." There is, of course, a long way to go. However, the combination of public health measures, decreasing smoking, and more widespread treatment of AAA in the elderly all appear to be combining to reduce rates of death from rAAA. With an the increasing elderly population and the fact much of the decrease in death from rAAA is due to elective repair in those aged ≥75 years, vascular surgeons can continue to contribute to the reduction in death from rAAA through increased safety of repair of AAA in the older population. Given the likely presence of higher levels of comorbidities in the elderly, continued improvement in deaths from rAAA will be a challenge for surgeons, anesthesiologists, intensivists, and primary care physician involved in the care of these elderly patients.

Mechanism of Ischemic Infarct in Spontaneous Cervical Artery Dissection

Morel A, Nagara O, Touze E, et al. Stroke 2012;43:14354-61.

Conclusion: In patients with cervical artery dissection (CAD), stroke is most frequently associated with direct and indirect signs of artery-to-artery embolization.

Summary: In young adults, extracranial CAD accounts for 20% of strokes (Leys D et al, Neurology 2002;59:26-33). It is somewhat controversial whether artery-to-artery embolization is the main mechanism of stroke in CAD or that stroke results from reduced flow from the primary cervical lesion. Should stroke result from reduced flow from the primary cervical lesion, one could argue for open or endovascular revascularization, whereas if artery-to-artery embolization is the primary event, anticoagulation or antiplatelet regimens would seem to be most advantageous to prevent secondary embolic events. Imaging studies can potentially distinguish hemodynamic from thromboembolic infarcts. For example, acute thromboembolism can be demonstrated directly on brain magnetic resonance (MR) imaging using the T2 sequence where intraluminal acute thrombus appears as signal loss along the course of an occluded symptomatic cerebral artery (Flacke S et al, Radiology 2000;215:476-82). Presumed embolic mechanism of stroke can also be evoked indirectly on diffusion-weighted imaging (DWI). DWIs demonstrating a pial or perforating territory stroke pattern are more likely reflective of an embolic mechanism of stroke; however, DWIs indicating junctional or watershed infarcts more likely indicate stroke of hemodynamic origin. The authors used imaging studies to identify the most likely mechanism of stroke in patients with CAD. They retrospectively evaluated cervical ultrasound studies, cervical MR angiography, and stroke brain MR imaging in consecutive patients with CAD. An embolic mechanism was considered as the mechanism stroke in cases of direct visualization of intracranial embolism as a susceptibility vessel sign on T2 imaging or in the case of pial artery territory infarction with DWI. A hemodynamic mechanism of infarction was implicated in the case of watershed infarction and pial artery territory infarction where two or more of the following were present: severe stenotic or occlusive CAD, reduced intracranial velocity on ultrasound or signal on MR angiography, or hyperintense vessel sign on fluid-attenuated inversion recovery. The remaining patients were considered to have a mixed mechanism of stroke after CAD. Of 172 consecutive patients with CAD, 100 (58%) had evidence of acute stroke on DWI imaging. Of these 100 patients, stroke was attributed to a thromboembolic mechanism in 85, to a hemodynamic mechanism in 12, and a mixed mechanism was felt to be present in only three.

Comment: The data in this study apply to patients with spontaneous CAD. Patients with CAD secondary to a traumatic mechanism do not appear to have been entered into the study. Nonetheless, the data do suggest that thromboembolism rather than hemodynamic infarction is the most frequent cause of stroke when a cervical artery dissects. Prevention of artery-to-artery embolization would therefore appear to be the major priority in the large majority of patients with CAD, certainly those with spontaneous CAD. The relative efficacy of antiplatelet therapy vs vitamin K antagonists in preventing artery-to-artery embolization in CAD is not definitely known. Randomized controlled trials are needed to obtain Level 1 evidence of the role of heparin or antiplatelet therapy and operative or endovascular therapies in the management of CAD.

Residential Proximity to Major Roadway and 10-Year All-Cause Mortality After Myocardial Infarction

Rosenbloom JI, Wilker EH, Mukamal KJ, et al. Circulation 2012;152:2197-203.

Conclusion: There is an increased risk of all cause 10-year mortality associated with living close to a major roadway at the time of acute myocardial infarction.

Summary: Living close to a major roadway results in increased exposure to traffic-related air pollution, traffic noise, and other potentially adverse health factors. Living close to a roadway has been associated with increased rates of coronary heart disease (Hoffmann B et al, Eur Heart J 2006;27:2696-702), increased stroke mortality (Maheswaran R et al, Stroke 2003;34:2776-80), and deep venous thrombosis (Baccarelli A et al, Circulation 2009;119:3118-24). Long-term exposure to air pollution is also known to be associated with increased cardiovascular mortality, possibility related to increased progression of atherosclerosis, increased inflammation, or increased oxidative stress. This study is one of the first to focus specificity on mortality associated to living close to a major roadway and is the first to address the relationship between living near major roadways and later mortality among survivors of an acute myocardial infarction. From 1989 to 1996, 3886 individuals were hospitalized for acute myocardial infarction in 64 centers in the United States. Patients living in institutions, those with only post office boxes, and those whose address could not be GEO coded were excluded, resulting in 3,547 for analysis. Distance to the nearest major roadway was assigned after addresses were GEO coded. Cox regression analysis was used to calculate hazard ratios, adjusting for personal characteristics, such as age, sex, race, education, and marital status, as well as the distance to nearest acute care hospital and clinical characteristic such as smoking, body mass index, comorbidities, and medications. Neighborhood characteristics were derived from the United States Census data (household income, education). In the patients studied, there were 1,071 deaths after 10 years of follow-up. In the fully adjusted model, compared with living >1000 meters from a major roadway, hazard ratios (95% confidence intervals) for a residence distance away from a major roadway were 1.27 (1.01-1.60) for <100 meters, 1.19 (0.93-1.60) for 100 to 200 meters, and 1.13 (0.99-1.30) for 200 to 1000 meters ($P = 0.016$ for trend).

Comment: The data indicate survival after acute myocardial infarction increases as one moves away from having a residence near a major roadway. This study adds to a growing consensus that living next to a major roadway, whether because of air pollution or other unknown factors, is associated with increasing manifestations of atherosclerosis. However, no study as of yet has specifically addressed proximity to roadways as a risk factor for peripheral arterial disease or other clinically evident manifestations of atherosclerosis.

Superficial and Deep Venous Thrombosis, Pulmonary Embolism and Subsequent Risk of Cancer

Sorensen HT, Svoerke C, Farkas DK, et al. Eur J Cancer 2012;48:586-93.

Conclusion: Lower limb deep venous thrombosis (DVT) and superficial venous thrombosis (SVT) are both preclinical markers of occult cancer, particularly during the first year after diagnosis.